Intracardiac Thrombus Causing Peritoneovenous Shunt Failure: Detection by Two-Dimensional Echocardiography

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Peritoneovenous shunts have become an accepted mode of therapy for ascites refractory to medical treatment. However, their use is known to be associated with significant morbidity and mortality. Reported is the case of a patient with a Denver shunt who developed massive intracardiac thrombosis and subsequent shunt malfunction, despite preserved shunt patency.

(J Am Coll Cardiol 1986;7:1174-6)

The development of peritoneovenous shunts in 1974 has improved the management of medically intractable ascites and may favorably influence the course of hepatorenal syndrome (1). Despite its beneficial effects, it remains controversial whether such shunting improves patient survival (2,3). Furthermore, the procedure is associated with frequent complications. Infection, shunt occlusion, disseminated intravascular coagulation and variceal hemorrhage have been reported after implantation of a peritoneovenous shunt (1–4).

We describe an unusual complication (right cardiac thrombus) after placement of a Denver peritoneovenous shunt (5), which was diagnosed by two-dimensional echocardiography.

Case Report

A 55 year old man with a 6 year history of ascites secondary to Laennec’s cirrhosis was admitted in December 1984 for evaluation of a malfunctioning Denver shunt. Between 1980 and this admission, management of the ascites included four consecutive peritoneovenous shunts. Two shunts were removed because of thrombotic shunt occlusion, and a third was removed after an episode of peritonitis with bacteremia. The last shunt was inserted through the right femoral vein in March 1984. Three weeks before admission, the patient noted “decreased suction” when activating the subcutaneous pump, together with worsening ascites and progressive leg edema. He denied having fever, chills, abdominal pain, chest pain or dyspnea.

On physical examination, vital signs were normal; however, there was pronounced jugular venous distension, tense ascites, a large umbilical hernia and edema of the scrotum and lower limbs. The cardiac examination revealed a widely split second heart sound that varied normally with the respiratory cycle. The relative intensity of the aortic and pulmonary components was normal.

Laboratory testing revealed a hematocrit of 33.1% and 58,000 platelets/mm³. The prothrombin time was 2 seconds longer than control values and the serum albumin was 1.9 g/dl; however, the bilirubin, alkaline phosphatase and hepatic transaminases were within normal limits. The creatinine was 2.7 mg/dl with normal serum electrolytes. Low QRS amplitude was present on the electrocardiogram.

Roentgenographic examination of the chest revealed normal heart size and pulmonary vasculature. The shunt was best visualized in the lateral projection, with its tip appearing to lie in the anterior third of the cardiac silhouette.

The central venous pressure was elevated to 21 cm H₂O, but arterial blood gases and pulmonary function tests were normal.

A radionuclide “shuntogram” was obtained on December 5 by injection of technetium-labeled macroaggregated albumin into the peritoneal cavity. The study showed little or no migration of the radionuclide to the lungs 5 minutes after the injection, but intense concentration of the tracer appeared within the shunt lumen 4.5 hours later. These findings were interpreted as indicating that the Denver shunt was patent, but that flow of ascites into the circulation was delayed.

A gated radionuclide ventriculogram revealed normal right and left ventricular ejection fractions.
Figure 1. Four chamber view of the two-dimensional echocardiogram. The right atrial (RA) portion of the mass (black arrow) probably consists of catheter and associated thrombus. The major portion of the right ventricular (RV) thrombus is beneath the white arrow. TV = tricuspid valve.

Two-dimensional echocardiography (Fig. 1). The left ventricle, left atrium and aortic root were normal, as were the left heart valves. The peritoneovenous shunt could be visualized in the inferior vena cava and in the right atrium. A linear structure crossing the tricuspid valve into the right ventricle was also noted in some frames. An echo-dense mass was seen within the right atrium, whereas a much larger mass filled most of the cavity of the right ventricle and appeared to be attached to its free wall. The patient was referred for thoracic surgery for removal of presumed intracardiac thrombi and repositioning of the shunt.

Surgical findings. The right atrium (Fig. 2) was almost entirely filled with thrombus, which extended across the tricuspid valve into the right ventricle, fixing the tricuspid valve in a partially closed position and thus creating tricuspid stenosis. The right atrial wall was hypertrophied, suggesting chronic tricuspid valve obstruction. The right ventricular cavity contained a large, irregular mass of thrombotic material which was adherent to the endocardium, chordae tendineae and papillary muscles, sparing only the outflow tract. Careful removal of the thrombus was performed; however, it was necessary to remove the anterior tricuspid valve leaflet. Microscopic examination of the thrombus revealed chronic and acute portions; the deeper layers were tightly adherent to the endocardium and included areas of dense fibrosis, necrosis and calcification. Bacteriologic analysis of the material failed to reveal bacteria or fungi.

Postoperatively, the patient developed renal failure requiring dialysis. A severe coagulopathy ensued, with further prolongation of the prothrombin time and marked thrombocytopenia. One week after surgery, the patient died from a massive gastrointestinal hemorrhage.

Discussion

Although thrombotic shunt occlusion is a relatively common cause of peritoneovenous shunt failure (6), extensive intracardiac thrombosis causing shunt malfunction without shunt occlusion has not been previously reported. In our patient, thrombosis in the right heart chambers may have resulted from the presence of an intracardiac foreign body, coupled with some degree of consumption coagulopathy associated with intravenous infusion of ascitic fluid. Normal

Figure 2. Intraoperative photograph demonstrating the right atrial portion of the mass (arrows) seen through an atriotomy. The heterogeneous composition of the thrombus is evident. RV = right ventricle.
right and left ventricular function, assessed by radionuclide ventriculography, excluded primary myocardial disease as the precipitant to thrombosis formation. The consequence of this extensive thrombosis was elevation of the right atrial pressure, resulting in loss of the normal pressure gradient between the peritoneal cavity and the right atrium. Poor shunt function without shunt occlusion thus occurred.

The most serious consequence of thrombi occurring in the large veins and in the right heart is pulmonary embolism. Fatal pulmonary emboli have been reported to originate from indwelling central venous catheters for total parenteral nutrition and from ventriculoatrial shunts for hydrocephalus (7–9). Thus, it is of the utmost importance to suspect and correctly diagnose this complication.

**Thrombus formation in peritoneovenous shunts.** Several factors predispose to thrombus formation in the presence of chronic indwelling venous catheters. Malposition of the tubing is one factor. The tubing may be insufficiently long, thereby lying in a relatively small vein; reduction of the effective vascular lumen and obstruction of blood flow result (6,10); alternatively, it may be too long and lie in the right ventricle or even in the pulmonary artery (6), where its excessive mobility predisposes to endothelial damage. Furthermore, insertion of the tubing into the inferior vena cava through a femoral vein, rather than into the supero vena cava through a subclavian or jugular vein (6), may promote thrombosis because of slower flow or higher venous pressure, or both. Infection (6,10), estrogen therapy (10), right ventricular failure (11) and a history of clotting of previous shunts (6) are other factors contributing to an increased thrombotic diathesis. In addition, endotoxins and procoagulants (12,13) derived from the cellular or humoral components of ascitic fluid represent further potential stimuli to thrombogenesis. Introduction of ascitic fluid into the circulation has been associated with the development of disseminated intravascular coagulation (3,13), which may resolve after shunt ligation without shunt removal. This coagulopathy appears to improve with early administration of aspirin and antplatelet agents (13).

**Echocardiographic diagnosis.** Two-dimensional echocardiography allows excellent, noninvasive visualization of the inferior vena cava and right heart chambers in multiple tomographic planes. Accurate localization and estimation of the size of intracardiac mass lesions is thus facilitated. Because its safety and sensitivity allow the clinician to avoid the potential hazards of cardiac catheterization in this setting, echocardiography is the diagnostic technique of choice when an intracardiac mass of any etiology is suspected (14–16).

The major sources of pulmonary emboli are thrombi originating in the deep veins of the leg and pelvis; several recent reports (14,15) suggest that such thrombi may become entrapped in the trabeculae or chordae of the right heart chambers where they are easily visualized by two-dimensional echocardiography (14). Thus, patients with pulmonary emboli in whom the source of embolism is not readily determined should have a two-dimensional echocardiographic examination of the right side of the heart. Earlier and more aggressive use of echocardiographic examination would also help to assess the frequency and the natural history of intracardiac thrombotic complications of indwelling catheters.

**In conclusion,** extensive intracardiac thrombosis may induce malfunction of peritoneovenous shunts in the absence of shunt occlusion. This complication is easily detected by two-dimensional echocardiography, and should be sought when the cause of shunt failure is not readily apparent by other diagnostic techniques.

**References**